Short report

Appearing and disappearing CT scan abnormalities in epilepsy in India—an enigma

B C BANSAL, ABHA DUA, RAKESH GUPTA, M S GUPTA

From the Department of Medicine and Division of Neurology and Department of Paediatrics, Medical College and Hospital, Rohtak, India

SUMMARY In 230 consecutive cases of epilepsy, CT abnormalities were found in 51.7%. Out of these, 91 cases (39.5%) had non specific abnormalities consisting mostly of ring lesions, hyperdense disc lesions with surrounding oedema in enhanced scans and in a small percentage hypodense lesions, generalised brain oedema and calcifications. All these cases were treated with anticonvulsant drugs alone. A follow up scan was possible in 31 cases, 12 weeks or later, after the control of the seizures. Out of these 31 cases, 24 showed a complete or significant resolution and five remained unchanged. Two of these cases showed an increase in the lesions which resolved on treatment with antituberculous drugs. These lesions therefore may have an aetiology other than tuberculosis in the majority of cases and there is ample justification in treating them initially with anticonvulsant drugs only.

CT abnormalities in cases of epilepsy have been variously reported between 36 and 56%,¹ 10,² and 60%.³ The abnormalities reported were tumours, trauma, infarcts, cerebral atrophy, hydrocephalus, porencephaly and vascular abnormalities.

Unusual CT abnormalities have been reported from India in various series consisting of ring and high attenuation disc lesions with surrounding oedema.⁴ These cases have been labelled as intracranial tuberculomas.^{4,5} The diagnosis was achieved through biopsy in a few cases but largely through the disappearance of these features in response to therapy with antituberculous drugs or circumstantial evidence on finding tuberculous lesions elsewhere in the body. It was later noticed that these lesions disappeared in some cases merely on treatment with anticonvulsants and without any antituberculous drugs.⁶ A contradictory report⁷ has come from India which again stressed the possibility of these lesions being tuberculomas. We report our experience of CT abnormalities in 230 consecutive patients with epilepsy who were willing to have a CT scan done in addition to other investigations.

Materials and methods

The study included 230 consecutive patients with epilepsy, focal and generalised, who attended the neurology clinic of the Medical College and Hospital, Rohtak, India, and who were willing to have a CT scan. Patients with syncope, hysterical seizures, blackouts of indeterminate nature, brain tumours, and exposure to intoxicants were excluded. Routine blood counts, ESR, urine, blood biochemistry-blood sugar, urea, chest and skull radiographs, EEG and cerebrospinal fluid (CSF) examinations were carried out in each case. CT was carried out within six weeks of an ictus and the majority were enhanced. CT was repeated after 12 weeks or more of anticonvulsant therapy wherever possible.

Observations and Results

A total of 230 cases were studied. Sixty three had focal and 167 generalised seizures. The age varied between five to 54 years (mean 23.58) and the male to female ratio was 2:1. Todd's palsy was seen in seven cases of focal epilepsy. The CT scan was abnormal in 119 (51.8%) cases. Twenty eight patients had a specific CT abnormality (cerebral atrophy 7, cysticercosis 8, gliosis 5, tumours 4, cystic lesions 2, arachnoid cyst and arterio-venous malformation, one each).

Ninety one cases had non-specific lesions enhancing ring lesions (25 cases; 27.5%), low attenuation (19 cases; 20.9%), and high attenuation disc lesions (20 cases; 22.0%). Both high attenuation and ring lesions had surrounding oedema. The other non-specific lesions seen were generalised brain

Correspondence to: Dr Bansal, 8/6J Medical Enclave, Rohtak, India 124001.

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Fig 1 (a) an enhancing ring lesion with surrounding oedema in a case of focal seizures. (b) showing complete resolution after treatment with anticonvulsants alone.

oedema (13 cases; 14.3%) and calcifications (14 cases; 15.4%). The focal CT lesions were seen even in generalised epilepsies without EEG correlates. In focal seizures, the CT lesions corresponded to the site of the focus. None of these patients had clinical or radiological evidence of tuberculosis anywhere in the body. CSF was normal in all cases. One case had a past history of left sided pleural effusion.

All these patients were put on anticonvulsant drugs only. A repeat CT scan was possible in 31 patients 12 weeks or more after control of the fits. The lesion resolved completely or significantly in 24 out of these 31 cases (77.4%) (fig). They remained unchanged in five (16.1%) and ring lesions increased in two (6.4%). One of these cases had a history of pleural effusion 10 years earlier. In addition both these patients were then given antituberculous drugs. A third scan after about 12 weeks showed marked resolution. Out of the five cases which remained unchanged one had a large low attenuation lesion which on biopsy showed only reactive gliosis. The follow up CT findings are summarised in the table.

Discussion

The pathogenesis of these lesions remains undetermined. Evidence suggests that tuberculosis was

present in only one case (out of 91) which had a history of left sided pleural effusion. Wadia *et al*⁷ argued that the resolution of these lesions without antituberculous drugs is no proof against tuberculosis as a spontaneous resolution of lesions in lungs is well described. It is, however, very difficult to believe that such a large percentage of symptomatic cases, where the seizures and CT lesions coincidentally appear and disappear without antituberculous drugs, could be spontaneously healing tubercular lesions. Some of these (two in this series) could be tuberculomas. We suggest, therefore, that these lesions should be watched for some time and that antituberculous drugs should only be started if the lesions persist. None of our 91 cases deteriorated clinically during the period of observation.

The pathogenesis of the CT lesions needs an alternative explanation. Gastaut⁸ reported a small sylvian infarct in patients with jacksonian seizures and prolonged neurological deficit. This was not likely in our patients as the lesions were not sylvian and the neurological deficit was absent or transient and the CT changes also spontaneously disappeared. Sethi *et al*⁶

Table Follow up scans ($n = 31$)

Type of lesion	Total	Significantly resolved	Completely resolved	Unresolved	Increased
Low attenuation	8	—	7†	1*	—
High attenuation	7	3	3	1	—
Ring	12	6	3†	1	2‡
Brain oedema	2	—	2	—	—
Calcification	2	—	—	2	—

*Biopsy performed on patient found to be reactive gliosis.

†One case resolved with calcified remnants.

‡One had a history of pleural effusion. Both showed complete resolution after addition of antituberculous drugs.

have suggested that they are due to a focal encephalitis peculiar to this region. To date there is no evidence to substantiate this theory. Jain and Ahuja⁹ reported a case of migraine with a low density area with enhancement on contrast injection which disappeared on follow up CT. A transient disruption of blood-brain barrier was suggested as a pathogenetic mechanism. This hypothesis also lacks laboratory and other evidence.

It has been suggested that these lesions could be the effect rather than the cause of a seizure. The enhancing ring lesions and high attenuation disc lesions are difficult to explain by this hypothesis. Moreover, all cases of seizures including those with status epilepticus do not have CT abnormalities.

Ultimately these lesions remain puzzling theories but since the lesions are benign all such patients should be observed on anticonvulsant drugs alone and submitted to serial CT.

References

- 1 Bognadoff BM, Stafford CR, Green L, *et al.* Computerised transaxial tomography in the evaluation of patients

- with focal epilepsy. *Neurology* (Minneapolis), 1975;**25**:1013–7.
- 2 Gastaut H, Gastaut JL. Computerised transverse axial tomography in epilepsy. *Epilepsia* 1976;**17**:325–36.
- 3 McGahan JP, Dublin AB, Hill PP. The evaluation of seizure disorders by computerised tomography. *J Neurosurg* 1979;**50**:328–32.
- 4 Bhargava S, Tandon PN. Intracranial tuberculomas; a CT study. *Br J Radiology* 1980;**53**:935–45.
- 5 Rao IM, Goulatia RK, Bhargava S. CT analysis of first 525 patients. *Neurology, India* 1980;**17**:86–94.
- 6 Sethi PK, Kumar BR, Madan VS, Mohan VS. Appearing and disappearing CT abnormalities and seizures. *J Neurol, Neurosurg Psychiatry* 1985;**48**:866–9.
- 7 Wadia RS, Makhale CN, Kelkar AN, Grant KB. Focal epilepsy in India with special reference to lesions showing ring or disc like enhancement on contrast computed tomography. *J Neurol, Neurosurg Psychiatry* 1987;**50**:1298–301.
- 8 Gastaut H, Boudouresques G, Gastaut JL, *et al.* Jacksonian epileptic seizures as inaugural manifestations of sylvian infarction revealed by computerised axial tomography. In: du Boulay GH, Moseley IF, eds. *The First European Seminar on Computerized Axial Tomography in Clinical Practice*. New York: Springer Verlag, 1977:237–42.
- 9 Jain S, Ahuja GK. Unusual transient high and low density CT lesions in Migraine: a case report. *Headache* 1986;**26**:19–21.